5-2019

Interactions Between an Education Polygenic Score and SES in Determining Health in Young Adulthood

James Bolyard

Interactions Between an Education Polygenic Score and SES in Determining Health in Young Adulthood *

Atticus Bolyard†

College of William & Mary

May 3, 2019

Senior Thesis in Department of Economics

*I would like to express my sincere gratitude for my advisor, Professor Peter Savelyev, for assistance in completing this thesis. Without his guidance, support, and patience, completing this work would certainly not be possible. I would also like to thank Peter McHenry and Elyas Bakhtiari for serving on my committee.

†E-mail: jabolyard@email.wm.edu

Abstract

I find that an education polygenic score (PGS) and socioeconomic status (SES) are positively associated with health in young adulthood. I also explore the moderating role of SES and contribute to the discussion on the casual status of education as a determinant of health.

1 Introduction

Growing availability of genetic data in recent years has allowed researchers to study gene-environment interactions with increasing statistical precision. Methodological and substantive advances have led many social and medical scientists to use polygenic scores (PGS), aggregate measures of genetic predisposition towards specific life outcomes. One type of score with particularly important policy implications is an education polygenic score, which measures an individual's genetic predisposition for completion of formal schooling. This paper shows that the same genetic mechanisms that are captured in an education polygenic score also predict favorable health outcomes. Additionally, this relationship is moderated by childhood socioeconomic status (SES); individuals who report experiencing particularly poor conditions in childhood do not experience the same beneficial health effects of the PGS as others who do not report experiencing these conditions.

The main contributions of this research are summarized as follows. First, I simultaneously model early skill and health formation, education choice, a health outcome, and unobserved heterogeneity to show that an education PGS is predictive of a variety of health outcomes. Second, I show that this genetic endowment exhibits differential effects depending on childhood SES.[1] Third, I contribute to a better understanding of the mechanisms through which the score works. Fourth, I also contribute to the debate on the causal status of the education-health relationship by ruling out important genetic confounders.

I use data from The National Longitudinal Study of Adolescent to Adult Health (Add Health), which follows a cohort of individuals who were in either middle or high school during 1996, and who are now young adults. I study a variety of health outcomes related

[1] In this paper, SES is a binary variable that takes the value of 1 if the respondent reported experiencing any one of three proxies for low SES in childhood: receiving government help, having trouble paying bills, or living in an unsafe neighborhood.

to general and mental health, substance use, exercise, and body weight.

Polygenic scores are powerful measures of genetic endowment. They may be thought of as the aggregate genetic endowment an individual has towards particular life outcomes. For instance, scores may be constructed from individual genetic data measuring an individual's propensity to smoke, to develop schizophrenia, or in this case, to complete additional years of formal education (Domingue et al., 2015). The effects of scores are not necessarily independent of environmental effects, and so using scores to understand gene-environment interactions is one promising line of research that is gaining traction across the social sciences (Domingue et al., 2015).

My most immediate and novel result is that the PGS I use–which is designed to predict years of formal education–also exhibits economically and statistically significant effects on multiple health outcomes. In particular, it exhibits a large effect on my main outcome of interest, which is general health. I show that these effects work not only through education, which is highly predictive of health, but also through channels independent of education. In other words, I find that the education PGS has a strong direct effect on health in addition to its indirect effect through education.

Previous work has suggested that education scores may contribute to health. Marioni et al. (2016) use data on European cohorts and show that an education PGS is predictive of parental longevity, and suggest that some genetic mechanism captured in childrens' education score might be relevant to determining parental health, since parents' genes are similar to their childrens'. However, Marioni et al.'s result may be related to better education of children leading to greater wealth and better parental care-giving, not necessarily inherited health endowments. It may also be confounded by the association between health and education: more educated parents may live longer. In contrast, I study the relationship between scores and health for the same individuals and find strong effects of the scores on health.

I am not the first to study the effect of Education PGS's on outcomes other than

education. Barth et al. (2017) find that a similar education score also explains large amounts of variation in stock market returns and wealth inequality. They offer suggestive evidence that the score may capture aspects of individual information-processing that affects decisions–such as conceptions of probability and risk aversion. This hypothesis is in line with my results. This model substantially broadens the set of affected outcomes by showing effects on essential health outcomes.

Health decisions follow specific decision-making processes and a considerable literature exists in economics and other disciplines that attempts to model this process (Grossman, 2017). Understanding the relative contributions of genes, education, and gene–environment interactions to health outcomes is crucial to understanding health formation. Additionally, decomposing the SES–health gradient has policy implications. This paper provides evidence that targeting low SES as a policy variable is effective at decreasing health inequality since it allows individuals to enjoy health benefits associated with their genetic endowment.

Family socioeconomic status (SES) affects a large variety of environmental influences that determine health, and so it is a natural candidate for a gene-environment study (Strauss and Thomas, 2007). Additionally, previous work has shown that SES moderates the effect of health-specific polygenic scores on particular health outcomes. Bierut et al. (2018) show that SES moderates the effect of a smoking PGS on actual smoking outcomes. Papageorge and Thom (2018) find that children from low-SES families experience smaller gains in education than children from high-SES families, conditional on their polygenic score. However, to the best of my knowledge, I am the first to study the interaction between an Education PGS and SES in models of health outcomes.

My model is similar to other models of gene-environment interaction in the literature, which usually involve including an interaction term between a polygenic score and some environmental influence in a regression model. However, I take this framework a step further by also estimating the indirect effect of the genetic score through potential

mediating pathways: actual education, latent skills, and early health. I then calculate two sets of marginal effects for each outcome for two levels of childhood SES (low and high). Since the exact genetic mechanisms captured in this score are not entirely known, decomposing the mechanisms through which the score works is informative for understanding this highly useful and increasingly popular genetic variable.

I simultaneously estimate a set of equations that model skills, early health, education, and health outcomes. I explicitly control for latent skills and for unobserved heterogeneity, which allows me to argue that if the model's assumptions are met, we may interpret effects as causal. It is a common concern in the literature that genetic scores are correlated with the effects of family environments or social conditions rather than pure genetic effects, since parents have similar genetic endowments as their children. This would confound the effects of any study using genetic scores. For instance, some aspects of intelligence, which I show is captured in the PGS, are heritable. Belsky et al. (2016) find that high-SES families tend to have higher scores. However, Lee et al. (2018) find that the education score still predicts educational attainment among siblings after controlling for family fixed effects. I aim to further minimize these concerns by explicitly controlling for a range of background variables, including siblings and birth order, family characteristics, and parental investment, as well as a number of social variables, including census tract-level crime, education, unemployment, and poverty rates. Additionally, since parental endowments often manifest in childrens' skills, controlling explicitly for skills diminishes concerns about this bias. Lastly, modeling unobserved heterogeneity should capture variation in individual family environments.

Fletcher (2019) uses variation in parental exposure to the 1918 influenza outbreak to show that parental exposure to poor environmental conditions moderate the effect of an Education PGS on actual education outcomes for daughters. Fletcher suggests that an "environmental bottleneck" exists that can limit the benefits of productive genetic endowments. By showing that low SES severely limits the positive health effect of an

education PGS, I provide additional evidence for the bottleneck hypothesis.

Skills are important determinants of both health-related outcomes and education. Economists have traditionally paid attention to skills that are malleable since they represent important policy inputs that can substantially impact crime, education, health, and labor outcomes (Heckman, 2008). They have also begun to pay increasing attention to noncognitive skills, and have shown that they are important determinants of health behaviors and outcomes (Conti et al. (2010); Heckman (2008); Savelyev and Tan (2017)). I examine whether the effects of the PGS on health work through skills.

Part of my results are surprising and negative, since they support neither the role of noncognitive skills nor the role of my measure of early health as mediators of the score. However, I do find small effects that work through education. This lack of effects may be due to the peculiar construction of the PGS, which only accounts for genes with particularly small p-values, a procedure which may neglect relevant genes with smaller effects, like those working through noncognitive skills.

My results may be compared to Belsky et al. (2016), who attempt to determine mechanisms through which a different education PGS affects education. I confirm their result that early health is not predicted by the score. I also confirm that the score is predictive of intelligence. However, while they find that the score is predictive of effects on early interpersonal skills and self-control, I do not confirm their result for Conscientiousness, Extraversion, or Emotional Stability. This may be because my model is conditional on a rich set of background controls including census data, household characteristics, and unobserved heterogeneity. While Belsky et al. (2016) offer an informative mediation model, effect sizes are small and a number of likely confounders are not controlled for.

My model also provides a contribution to a separate body of literature in health economics. Although economists have thoroughly documented the positive association between educational achievement and beneficial health outcomes, the causal status of this association is still debated (e.g., Galama et al. (2018)). One possible confounding

factor in education-health studies is genetic endowment: perhaps the same factors that lead individuals to educational attainment–preferences and skills that are based in genetic difference–also lead to beneficial health decisions (Boardman et al., 2015). I find that even after controlling for the education polygenic score, among other controls including latent skills and unobserved heterogeneity, actual education still exhibits large and statistically significant effects on a variety of health outcomes. Ruling out a potential confounder strengthens the evidence that education has a causal effect on health.

2 Data

I use data from waves I-IV of the National Longitudinal Study of Adolescent to Adult Health. This panel dataset follows roughly 20,000 individuals and contains detailed information on their family background, polygenic scores, health outcomes, skills, and education. The respondents were first surveyed in 1995-1996, when they were in grades 7–12, and were followed into adulthood. The most recent data, Wave IV, were collected when participants were 24–32 years old.

My sample size is constrained by the availability of genetic data. About 9,000 Add Health study participants took part in genotyping, 5,728 of whom are white.

Since gene expression varies by geographic ancestry, I perform this analysis only for individuals who self-identify as white.[2] I pool male and female respondents to increase power.

Table 1: Health, Skills, Education, and Genetic Data Description

	Males			Females			Pooled		
	Average	Standard deviation	Sample size	Average	Standard deviation	Sample size	Average	Standard deviation	Sample size
Education									
High School Dropout	0.050	0.218	1,802	0.042	0.201	2,011	0.046	0.209	3,813
Bachelor's degree	0.287	0.452	1,802	0.346	0.476	2,011	0.318	0.466	3,813
Genetic Scores									
Education Polygenic Score (a)	0.055	1.003	1,802	0.000	0.979	2,011	0.026	0.991	3,813
Principal Component 1	-0.001	0.399	1,802	0.004	0.468	2,011	0.001	0.437	3,813
Principal Component 2	0.007	0.482	1,802	-0.001	0.747	2,011	0.003	0.636	3,813
Principal Component 3	0.002	0.661	1,802	-0.016	0.694	2,011	-0.008	0.679	3,813
Principal Component 4	-0.011	0.761	1,802	0.010	0.678	2,011	0.000	0.719	3,813
Principal Component 5	0.028	0.670	1,802	0.015	0.690	2,011	0.021	0.681	3,813
Skills and Early Health									
General Health, Wave I (a)	0.126	0.949	1,802	-0.030	0.936	2,011	0.044	0.945	3,813
Conscientiousness (a)	0	1	1,792	0	1	1,991	0	1	3,783
Extraversion (a)	0	1	1,628	0	1	1,891	0	1	3,519
Emotional Stability (a)	0	1	1,799	0	1	2,009	0	1	3,808
Intelligence (a)	0	1	1,320	0	1	1,538	0	1	2,858
Health Outcomes									
General Health (a)	3.754	0.870	1,802	3.714	0.906	2,011	3.732	0.889	3,813
Obesity	0.351	0.477	1,779	0.339	0.473	1,989	0.344	0.475	3,768
No Exercise	0.120	0.325	1,721	0.151	0.358	1,933	0.136	0.343	3,654
Heavy Drinking	0.313	0.464	1,791	0.212	0.409	2,004	0.259	0.438	3,795
Smoking	0.426	0.495	1,794	0.367	0.482	2,006	0.395	0.489	3,800
Marijuana Use	0.104	0.305	1,800	0.052	0.223	2,010	0.077	0.266	3,810
Depression	0.128	0.334	1,802	0.250	0.433	2,011	0.192	0.394	3,813
Suicidal Thoughts	0.074	0.262	1,794	0.073	0.260	2,010	0.073	0.261	3,804

Notes: (a) indicates that the variable was standardized prior to analysis.

2.1 Education and Health Outcomes

I study two education thresholds: high school completion and college completion. Previous work on the returns to education has shown that having a Bachelor's degree is particularly predictive of health (Buckles et al., 2016). Distinguishing between high school dropouts and high school graduates allows us to quantify health effects of the PGS for individuals through an additional education margin. There is extensive evidence that high school graduation has substantial health benefits (Freudenberg and Ruglis, 2007).

All of the health outcomes in this study are measured in Wave IV, when participants were 24–32 years old.

My main health outcome of interest is self-rated general health. Respondents rated their health on a qualitative scale with five options (excellent, very good, good, fair, poor). The score was normalized for the estimation sample, and is treated as a continuous outcome. Self-reported health has been shown to be predictive of mortality, and is an essential measure of overall health (Idler and Benyamini, 1997).

I investigate three dummy variables related to substance use: heavy marijuana use into adulthood, smoking tobacco, and heavy drinking. Marijuana use is defined as smoking marijuana more than one or two times a week, on average, during the last year. Smoking is defined as smoking cigarettes at least once within the past 30 days. Heavy drinking is defined as consuming 4 or more drinks per drinking occasion as a female, and 5 or more drinks per occasion as a male, which follows the Centers for Disease Control and Prevention's definition of binge drinking. A limitation of this measure is that it does not account for frequency of drinking behavior.

I also analyze two lifestyle variables (obesity and no exercise) and two mental health variables (having suicidal thoughts and depression). Obesity is defined according to the

[2]Self-reported race does not always correspond to a distinct genetic ancestry, and so the functions of genes can vary substantially even within groups that identify as the same race. To account for this, I also control for the first five principal components of ancestry provided by Add Health.

usual Body Mass Index definition which is used by the National Institute of Health (BMI $\geq$ 30). No exercise is defined as one if the respondent reports not playing any sports, exercising outside, walking for exercise, or engaging in other physical activity during the past week.[3] The suicidal thoughts variable is set to one if the respondent reported seriously considering suicide within the last year. Depression takes the value of one if the respondent had ever been told by a health care provider that she has depression.

For summary statistics, see Table 1.

2.2 Polygenic Scores

Polygenic scores are measures of an individual's genetic predisposition to exhibit a particular phenotype. Although the effect of the genetic endowment is contingent on environmental factors–such as family characteristics and educational institutions–scores are still predictive across environment, although some heterogeneity in effect sizes exists. I use a score constructed by Lee et al. (2018), and based on a sample of 1.1 million individuals. The score predicts about 11 % of variation in years of education completed among a validation sample that includes Add Health data (Lee et al., 2018). Controlling for within-family effects may attenuate the effect of the score by around 40%, but does not eliminate it. The education score is standardized before analysis.

A major methodological concern is that scores may exhibit bias, or provide decreased statistical power, if population stratification isn't controlled for, since the functions of genes vary by anthropological background (Price et al., 2006). To address this, my study is based only on those who self-report being "white", since this is the most represented racial group in Add Health, and since the education score I use is constructed from a sample of individuals with European ancestry. Additionally, I control for principal components of genetic ancestry, which is a robust method for controlling for population

[3]Respondents were asked whether they participated in a wide range of physical activities, and No Exercise was coded as zero if they engaged in any. For the complete list, see questions H4DA2–H4DA9 of the Add Health Codebook.

stratification (Price et al., 2006).[4]

Scores are constructed by regressing a large number of individual genes (single nucleotide polymorphisms, or SNP's) on an outcome of interest, and, given the high risk of false-positives, keeping only coefficients with very low p-values, which will then be used to weight the contribution of each single gene to the overall score. For each individual, a score is constructed by creating a weighted sum all of the relevant genes an individual possess (Domingue et al., 2015).[5] The score used in this paper is constructed based on individual genes' contribution to years of completed schooling.

2.3 SES

I create a dummy variable indicating whether an individual reports experiencing at least one of three situations that are indicative of a particularly low family-SES: whether their family was on government assistance (such as welfare), whether their family had difficulty paying bills, and whether they lived in an unsafe neighborhood. 31.9% of the sample has low SES according to this definition.

2.4 Noncognitive Skills and Intelligence

Although multiple schema exist for representing noncognitive skills, one robust representation is the Big Five Personality taxonomy, which is grounded in personality psychology. Most noncognitive traits map into the Big Five in some manner (Borghans et al., 2008). The Big Five Skills are Openness, Conscientiousness, Extraversion, Agreeableness, and Neuroticism. Since skills are malleable over the lifecycle (Borghans et al., 2008; Fletcher and Schurer, 2017), I use only skills measured during the first wave of the

[4]All of my models are conditional on principal components of genetic ancestry, which are constructed from genetic data in the Add Health samplyee (Harris and Braudt, 2018).

[5]The score used in this paper is constructed from an analysis over about 1.1 million individuals of European descent, and identified 1,271 individual genes that are predictive of years of education (Lee et al., 2018).

Add Health study to avoid issues associated with reverse causality.

I use factor analysis to estimate factor scores of skills. Due to data limitations, I am not able to study Agreeableness and Openness, but I do construct scores measuring early Conscientiousness, Extraversion, Neuroticism, and Intelligence.[6] I construct factor scores for these traits from measures obtained when the Add Health cohort was in grades 7–12. I used exactly the same measures as proposed by psychologists using Add Health data, and verify that this provides a well-specific model (Young and Beaujean, 2011).[7] I also use three measures to construct factor scores for cognition. Since aspects of skills are genetic (Borghans et al., 2008), I investigate whether the effects of the PGS work through skills.

I use participant scores on the Add Health Picture Vocabulary Test, recent science grades, and recent math grades as measures for intelligence. The Add Health Picture Vocabulary test is a shortened version of the Peabody Picture Vocabulary Test.

2.5 Controls for Family Background, Geographic Information, and Initial Health

I control for a range of background variables that could influence education and health decisions. These include dummy variables indicating whether the respondent is hispanic; if the respondent's parents are college-educated; that cigarettes were smoked in the respondent's household; whether the respondent is an only child, a first born, a second born, or a third born child; if the respondent had an abnormally low birth-weight; whether the respondent lived in a rural, urban, or suburban area during Wave I; the

[6] In the rest of the paper, I will refer to Emotional Stability, which is the opposite of Neuroticism. Reversing the sign of this score does not change any substantive result, but aids interpretation.

[7] Young and Beaujean (2011) find that Conscientiousness, Extraversion, and Emotional Stability (Neuroticism) can be reliably constructed from Wave I questions. Although more reliable measures of the Big Five are available in Wave IV, I use the earliest measures available since skills change over time.

Table 2: Control and SES Measure Description

	Males		Females		Pooled	
	Average	Standard Deviation	Average	Standard Deviation	Average	Standard Deviation
Low SES Measures						
Unsafe neighborhood	0.064	0.245	0.064	0.245	0.064	0.245
Household received assistance	0.192	0.394	0.226	0.418	0.210	0.407
Trouble Paying Bills	0.114	0.318	0.128	0.334	0.121	0.327
Low SES Dummy	0.302	0.459	0.334	0.472	0.319	0.466
Family and Demographic Background						
Hispanic	0.069	0.254	0.054	0.226	0.061	0.240
Has a College-Educated Parent	0.548	0.498	0.497	0.500	0.521	0.500
Cigarettes smoked in house	0.457	0.498	0.483	0.500	0.471	0.499
Family income (log)	3.720	0.746	3.691	0.775	3.705	0.762
Only child	0.046	0.210	0.045	0.207	0.045	0.208
First born	0.321	0.467	0.317	0.466	0.319	0.466
Second born	0.321	0.467	0.296	0.457	0.308	0.462
Third born	0.117	0.322	0.117	0.322	0.117	0.322
Number of Siblings	2.456	1.887	2.631	1.987	2.548	1.942
Low Birth Weight	0.079	0.270	0.091	0.288	0.086	0.280
Rural	0.362	0.481	0.368	0.482	0.365	0.482
Suburban	0.404	0.491	0.382	0.486	0.392	0.488
Urban	0.218	0.413	0.228	0.420	0.223	0.417
Parents married	0.802	0.398	0.790	0.407	0.796	0.403
Evening meals with parents	5.041	2.242	4.938	2.363	4.987	2.307
South	0.371	0.483	0.354	0.478	0.362	0.481
Northeast	0.150	0.358	0.158	0.364	0.154	0.361
West	0.141	0.348	0.148	0.355	0.145	0.352
Midwest	0.338	0.473	0.341	0.474	0.339	0.474
Age Wave I: 10-12	0.080	0.271	0.113	0.317	0.098	0.297
Age Wave I: 13-14	0.303	0.460	0.345	0.476	0.325	0.469
Age Wave I: 15-16	0.402	0.491	0.368	0.482	0.384	0.486
Age Wave I: 17-19	0.208	0.406	0.171	0.377	0.189	0.391
Census Regional Variables						
Crime rate (per 100,000)	4657.295	2508.547	4783.954	2566.316	4723.943	2539.551
Education rate	0.228	0.130	0.227	0.130	0.227	0.130
Unemployment rate	0.064	0.024	0.065	0.024	0.064	0.024
Poverty rate	0.114	0.091	0.116	0.091	0.115	0.091
Biological Sex						
Male	1	0	0	0	0.473	0.499
Sample Size	1,802		2,011		3,813	

Notes: The reported sample size is the maximum of all variables reported in this table. This descriptive table is based on data before imputation.

respondent's region at Wave I (West, Midwest, South, or Northeast); and whether the respondent's parents were married at Wave I. I control for the following continuous variables: the log of family income; the respondent's number of siblings; and the number of evening meals the respondent eats with her parents each week. Additionally, I control for several tract-level census variables: the crime rate, education rate (defined as percentage of adults with a college degree), unemployment rate, and poverty rate of the respondent's census-tract. Together, these variables capture a large amount of information about the respondent's family and social environment that minimizes concerns about an upward bias of genetic effects. Lastly, I control for biological sex (male or female), as reported in Wave I. See Table 1.

In addition, I control for general health as reported by respondents during Wave I. This variable is constructed from earlier answers to the exact same question that I use for the main outcome variable, general health from Wave IV.

3 Methods

I use confirmatory factor analysis to analyze the choice of measures of intelligence (G) and Young and Beaujean's (2011) proposed measures of Conscientiousness (C), Extraversion (E), and Emotional Stability (ES). [8] Factor scores provide estimates of latent variables that can be used for statistical inference when properly estimated (Anderson and Rubin, 1956).

The factor models are specified as follows, where Θ^s is a latent skill such that $s \in \{C, E, ES, G\}$ and M_k^s is the k-th dedicated measure of skill s ($k \in \{1, 2, 3\}$):

$$M_k^s = \beta_{0,k}^s + \beta_{1,k}^s \Theta^s + \epsilon_k^s, \tag{1}$$

[8] My specification's RMSEA is 0.059 and CFI is 0.897. CFA was performed for a sample of 6500 individuals to maximize power. Generally, CFI should be above .95 and RMSEA should be below .08 (Cangur and Ercan, 2015), which are close to my fit measures.

where $\beta^s_{0,k}$ is the intercept, $\beta^s_{1,k}$ is the factor loading, and ϵ^s_k is the error term. I make the standard assumption that $\epsilon^{s_1}_k \perp\!\!\!\perp \epsilon^{s_2}_j, k \neq j$, for any $s_1, s_2 \in \{C, E, ES, G\}$, and error terms are independent of factors. Each latent variable is normalized to have mean zero and to be a positive representation of the skill.[9] From this factor model I calculate Bartlett factor scores $\boldsymbol{\Theta}$. Bartlett's method provides unbiased factor score estimates based on maximum likelihood techniques (Yung and Yuan, 2013).

I simultaneously estimate a model of initial skill and health formation, education choices, and a health outcome, using a full maximum likelihood estimation technique. I take advantage of the sequential nature of early skill and initial health formation, schooling decisions, and health outcomes in adulthood. Skills and initial health are formed before schooling decisions are made, which are formed before adult health outcomes are measured. These exclusion restrictions help identify the model (e.g., Cameron and Trivedi (2005)). Simultaneously estimating a system of linear equations provides both gains in efficiency and allows us to control for unobserved heterogeneity. I control for unobserved heterogeneity using the semi-parametric heterogenity model (Heckman and Singer, 1984).[10] I interpret this control for unobserved heterogeneity as capturing important personal, family, or genetic characteristics outside of what is explicitly controlled for.

Each model contains the set of equations described below, using the following notation: H_i, health outcome of interest in young adulthood; D_l, education threshold; G, education PGS; S the dummy variable indicating low-socioeconomic status; $G \cdot S$, the PGS-SES interaction (where PGS is standardized for the estimation sample); $\mathbf{X}$, the vector of controls; $\boldsymbol{\Theta}$, the vector of factor scores; H_0, early health; D, the vector of education outcomes; μ, unobserved heterogeneity that is constant across equations; and ϵ, idiosyn-

[9] For instance, Emotional Stability always implies more stability, not less.

[10] The model is also known as latent class analysis (Aitken and Rubin, 1985), discrete factor approximation (Mroz, 1999), and finite mixture modeling of unobserved heterogeneity (Cameron and Trivedi, 2005).

cratic error terms. Due to computational limitations, I estimate the model separately for each health outcome H_i.

$$H_0 = a_0 + a_G G + a_S S + a_{GS} G \cdot S + a_X X + \mu_{H_0} + \epsilon_{H_0} \tag{2}$$

$$\Theta^s = b_0 + b_G G + b_S S + b_{GS} G \cdot S + b_X X + \mu_{\Theta^s} + \epsilon_{\Theta^s} \tag{3}$$

$$s \in \{C, E, ES, G\}$$

$$D_l = c_0 + c_G G + c_S S + c_{GS} G \cdot S + c_X X + c_\Theta \Theta + c_{H_0} H_0 + \mu_{D_l} + \epsilon_{D_l} \tag{4}$$

$$l \in \{1, 2\}$$

$$H_i = d_0 + d_G G + d_S S + d_{GS} G \cdot S + d_X X + \beta_\Theta \Theta + d_{H_0} H_0 + d_D D + \mu_{H_i} + \epsilon_{H_i} \tag{5}$$

$$i \in \{1, 2, 3, 4, 5, 6, 7, 8\}$$

I impute missing control variables and factor scores to increase sample size. Using the MCMC procedure preserves the variance-covariance matrix of variables (Schafer, 1999).

4 Results

There are three main results of this work, beyond the main result that the education PGS is predictive of health. First, I show that, in a naive regression that does not control for actual education or a complete set of background controls, a dummy capturing particularly low levels of SES is more predictive of health outcomes than overall family income. The interaction between this dummy and the education PGS is also more predictive than the interaction between income and the PGS. This result suggests that there is some non-linearity in the effects of SES on health when interacted with the PGS: there appear to be stronger interaction effects only for individuals with comparatively lower family SES levels.

Second, I further investigate the relationship between the education PGS and health outcomes by controlling for a greater suite of background variables, actual education, unobserved heterogenity, and skills. I jointly estimate this model with a system of equations predicting the effect of the PGS and controls on potential mediators of the PGS-health relationship. I then decompose the overall marginal effect of PGS on health into health, skills, education, and direct components for both poor–SES ($SES = 1$) and high–SES individuals ($SES = 0$). I find that the polygenic education score is much more predictive of both overall health and various health outcomes for individuals who did not experience particularly low SES levels in childhood.

Lastly, my work contributes to the debate on the causal effect of education on health. Although the effect of education on health, longevity, and health mechanisms is extensively documented, the causal status of this relationship is an ongoing area of research. A plausible explanation for the observed relationship that undermines the causal effect claim is that the effect of education on health is actually capturing some underlying genetic ability or preference that is productive for both education and health. I show that this is plausible, since the education polygenic score is also predictive of health. However, despite controlling for this score I also find effects of education on health that are both economically and statistically significant. This suggests that some aspect of education is productive of health, even after controlling for genetic endowment.

Every health outcome I study, with the exception of general health, is a binary variable. Since I estimate models of these outcomes using logit models, I report marginal effects at the average instead of coefficients. These may be interpreted as the effect of an increase in one standard deviation of the polygenic score.

4.1 Nonlinear Effects of the PGS-SES Interaction

I start with two versions of a simple reduced-form model to investigate the interaction effect between the PGS and SES. Unlike my main model, these simple models do not

control for early skill formation, achieved education, or the majority of my background controls. In the first simple model, I interact the Education PGS with the individual's family income. In the second model, I interact the score with the SES dummy variable. I find that although both models show some interaction with childhood SES in predicting health outcomes, the model using the dummy variable is predictive of health across a larger range of outcomes and leads to more precisely determined results. This suggests that the interaction between the score and childhood SES is non-linear: it is much stronger when modeling the effects of a particularly low SES, as opposed to modeling overall income. The effects in both models show that poor SES is predictive of adverse health outcomes and that the PGS is predictive of beneficial health outcomes. They also show that the interaction term tends to diminish the effect of PGS for those with low SES. In other words, the health benefits of a higher PGS are canceled by low-childhood SES.

My simple model 3 is similar to Bierut et al. (2018), who show that SES moderates the effect of a polygenic risk score associated with smoking on peak-cigarette consumption. The authors construct an ordinal measure of childhood socioeconomic status, taking on a value from 0–3, that includes information on father's unemployment, whether the family ever moved or asked for help, and whether the family reports being financially well-off. My definition differs from this in that I do not assume that such a variable exhibits linear effects. Additionally, since I am interested in capturing the unique effects of individuals who grew up on the low end of the SES spectrum, I use slightly different SES measures. However, my result–that childhood SES moderates the effects of a polygenic score–complement Bierut et al.'s. While they find that high-childhood SES dampens the negative effect of a smoking PGS on smoking, I find that low-childhood SES dampens the positive effect of an education PGS on health-related outcomes.

I find that the education polygenic score, when interacted with the "poor childhood socioeconomic status" dummy variable, exhibits a substantial effect on general health.

However, when the same model is estimated using the log of childhood family income instead, which should theoretically capture similar information as the SES dummy, the effect disappears. I also find that the PGS-SES interaction is more predictive of plausible health mechanisms than the PGS-income interaction. Lastly, the poor SES dummy has uniformly larger marginal effects, and is more predictive of general health and plausible health mechanisms than family income. See Table 3.

In particular, I find that both poor SES and childhood income exhibit statistically significant marginal effects on the following outcomes: general health, heavy drinking, smoking, obesity, no exercise, and suicidal thoughts. Additionally, poor SES is directly predictive of marijuana use and depression at the means of the explanatory variables; the marginal effect of income on these outcomes is not precisely determined. In all cases, the effects work in the expected directions: poor SES is associated with worse health outcomes, and a higher family income is associated with better health outcomes.

In both models, a higher education polygenic score is predictive of a higher general health rating, and a lower likelihood of heavy drinking, smoking tobacco, marijuana use, obesity, and depression. In the model using the SES dummy, but not in the model using income, a higher polygenic score also predicts a lower likelihood of no exercise. Neither model shows an effect of the PGS on suicidal thoughts.

The interaction effects also vary between the two models. In the model using income, only one income x PGS effects is precisely determined, at the 10% level: the interaction is health-protective, and reduces the likelihood of marijuana use. Four interaction terms are significant in the model using SES; SES x PGS predicts general health (5% level), marijuana use (5% level), no exercise (10% level), and depression (10% level). The interaction effects for the SES model are harmful to health. They may therefore be interpreted either as poor social position "canceling" the protective effect of the polygenic score, or as poor genetic endowment "canceling" the positive effects of a favorable social position. Alternatively, combinations of high social position and a high PGS, or low social position and

a low PGS, may be considered uniquely more protective/harmful than the sum of their direct effects, respectively. In the more complex model, I decompose marginal effects by channel to better understand the mechanisms behind these effects. I also control for a larger set of controls, education, and unobserved heterogeneity.

This result mirrors the effects found in a similar study which examined the way in which SES moderates the effect of the education polygenic score on education outcomes. Papageorge and Thom (2018) found that the relationship between education and the PGS is much stronger for individuals born into richer families, and suggest that nurturing home environments and genetic endowments are substitutes for preventing particularly bad education outcomes, and complements in producing particularly good education outcomes. My results suggest that this may hold for health outcomes as well.

4.2 Decomposition of the Marginal Effect of the PGS on Health

If the score were predictive of education and only education, then we would expect the direct effect of the score on health outcomes to be zero, after controlling for actual education. The fact that the estimated direct effect is large suggests that genes predicting schooling also predict health.

I find that for individuals who did not experience low SES in childhood, the education polygenic score exhibits a statistically and economically significant total marginal effect on general health (8.1 %), heavy drinking(-1.8 %), smoking tobacco (-5.0 %), obesity (-1.7 %), marijuana use (-1.5 %), no exercise (-1.4 %), and depression (-2.6 %). For individuals who did experience low SES in childhood, the polygenic score only exhibits a statistically significant marginal effect on smoking tobacco (-6.6 %). All results are protective of health. See Table 4.

This total marginal effect can be decomposed into an indirect effect, which works through education, and a direct effect. The direct effect can be further broken down into the effect of the PGS through early health, cognitive and noncognitive skills, and an un-

Table 3: Associations between Health-Related Outcomes and Gene-Environment Interactions

	Health	Heavy Drinking	Smoking Tobacco	Marijuana Use	Obese	No Exercise	Depression	Suicidal Thoughts
A) Interaction with SES								
Education Polygenic Score	0.109 ***	-0.027 ***	-0.048 ***	-0.018 ***	-0.031 ***	-0.019 ***	-0.025 ***	0.004
	0.019	0.009	0.010	0.005	0.010	0.007	0.008	0.005
Poor SES	-0.278 ***	0.037 **	0.069 ***	0.028 ***	0.067 ***	0.030 **	0.027 **	0.029 ***
	0.035	0.015	0.018	0.008	0.017	0.012	0.013	0.008
Education PGS x Poor SES	-0.082 **	0.022	-0.005	0.018 **	0.003	0.023 *	0.026 *	-0.008
	0.035	0.015	0.018	0.008	0.017	0.012	0.013	0.008
Sample Size	3813	3795	3813	3810	3768	3654	3813	3804
B) Interaction with Income								
Education Polygenic Score	0.074 ***	-0.022 ***	-0.051 ***	-0.012 ***	-0.020 **	-0.008	-0.019 ***	0.000
	0.017	0.008	0.009	0.004	0.008	0.006	0.007	0.004
Log of Family Income	0.121 ***	-0.017 **	-0.027 ***	-0.007	-0.041 ***	-0.012 **	0.003	-0.008 *
	0.017	0.007	0.009	0.004	0.008	0.006	0.007	0.004
Edu PGS x ln (family income)	-0.007	-0.012	-0.002	-0.007 *	-0.001	-0.007	-0.009	-0.002
	0.016	0.007	0.009	0.004	0.008	0.006	0.007	0.004
Sample Size	3454	3439	3454	3451	3414	3311	3454	3445

Notes: All results are conditional on a limited set of controls: Principal Components 1–5 for ancestry, dummies for US region, cohort, and gender. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level.

observed channel. The direct effect is consistently larger in magnitude than the indirect effect across health outcomes. For high-childhood SES individuals, the magnitude of the effect of the polygenic score through education is at most 1.9% (on smoking tobacco), while it is at most 2.9 % (on general health) for the low-childhood SES individuals. By contrast, the magnitude of the direct effect for high-childhood SES individuals is as high as 6.4 % (on general health) and for low-childhood SES individuals is as high as 4.7 % (on smoking). In addition, all statistically significant direct effects contribute to a statistically significant total effect. In some cases, a statistically significant, but small, indirect effect and a statistically insignificant direct effect combine to produce a statistically significant overall effect of the PGS: this is true for high-childhood SES individuals' heavy drinking, obese, and no exercise outcomes.

The decomposition of the direct effects is shown in Table 7. The majority of these effects are driven by a large unobserved channel. The polygenic score may be capturing preferences, skills, or decision-making processes that are not explicitly modeled, and that generate both education and productive health outcomes. The unobserved marginal effect is significant for general health (5.2%), smoking tobacco (-2.6%), marijuana use (-1.0 %) and depression (-1.9 %), for high-SES individuals. For low SES individuals, only the unobserved marginal effect for smoking is significant (-4.0%). For high-SES individuals, the marginal effect of the PGS through skills is statistically significant for three health outcomes (health, smoking tobacco, and heavy drinking), but economically insignificant. No such effects exist for low SES individuals. I find that the effects of the PGS through early health is either statistically or economically insignificant, with the exception of the general health outcome for low-SES individuals (1%).

Together with the results of Barth et al. (2017), this paper suggests that genetic variants associated with education are pleiotropic, meaning that they are predictive of more than one phenotype. While I speculate that similar decision-making processes, which vary with genetic background, are involved in making education, health, and finan-

cial decisions, this model is not fine-grained enough to show where exactly pleiotropy occurs. For example, the genes captured by the score may affect extremely basic neurological functions that cannot be described using tools from decision theory.[11]

The polygenic score does not appear to work through early health or observed noncognitive skills. Table 5 shows that the score is related to IQ, however. The PGS-SES interaction is not predictive of any these early-life outcomes.

The indirect marginal effects, which work through education, are decomposed into effects that work through high school dropout and effects that work through college education. I do not observe any statistically significant marginal effects that work through high school dropout for either SES group, beyond a few very small effects ($\leq$ .4%). For bachelor's degree, I observe small, but significant health-protective marginal effects for the high-SES group on every outcome except for marijuana use (effects range in magnitude from .4% to 1.6%). For the low-SES group I observe small significant effects for general health and smoking tobacco through Bachelor's (2.9% and -1.7%, respectively, in addition to two economically insignificant results.[12]

I find that the PGS, SES, skills, and early health all affect education (see Table 6). Interestingly, I do not find a significant marginal effect of the PGS on being a high school dropout, although it does predict Bachelor's degree (2.8 %). Since only IQ is generated by the PGS, I do not find a large effect of PGS on health outcomes through education, despite the effects that early health and noncognitive skills have on education (see Table 6).

[11]For an overview of the mechanisms through which observed pleiotropy may occur, see Solovieff et al. (2013).

[12]For a complete decomposition of the mechanisms through which the score works through education, see the Appendix.

Table 4: Aggregated Direct and Indirect Effects of the PGS on Health Outcomes

	Health (1)	Heavy Drinking (2)	Smoking Tobacco (3)	Marijuana Use (4)	Obese (5)	No Exercise (6)	Depression (7)	Suicidial Thoughts (8)
High SES								
Indirect effect	0.017 ***	-0.007 ***	-0.019 ***	-0.005 ***	-0.005 **	-0.004 ***	-0.005 ***	0.000
	(0.004)	*(0.002)*	*(0.003)*	*(0.001)*	*(0.002)*	*(0.001)*	*(0.002)*	*(0.001)*
Direct effect	0.064 ***	-0.011	-0.031 ***	-0.010 **	-0.014	-0.010	-0.020 ***	0.004
	(0.019)	*(0.009)*	*(0.011)*	*(0.004)*	*(0.010)*	*(0.007)*	*(0.008)*	*(0.004)*
Total	0.081 ***	-0.018 **	-0.050 ***	-0.015 ***	-0.019 *	-0.014 **	-0.026 ***	0.004
	(0.019)	*(0.009)*	*(0.011)*	*(0.004)*	*(0.010)*	*(0.007)*	*(0.008)*	*(0.004)*
Low SES								
Indirect effect	0.029 ***	-0.006 *	-0.019 ***	-0.005 **	-0.003	-0.006 **	0.001	-0.001
	(0.009)	*(0.003)*	*(0.005)*	*(0.002)*	*(0.004)*	*(0.003)*	*(0.003)*	*(0.002)*
Direct effect	-0.022	0.004	-0.047 ***	0.006	-0.019	0.010	-0.003	-0.004
	(0.031)	*(0.013)*	*(0.016)*	*(0.005)*	*(0.014)*	*(0.009)*	*(0.010)*	*(0.006)*
Total	0.006	-0.002	-0.066 ***	0.001	-0.022	0.003	-0.002	-0.005
	(0.030)	*(0.013)*	*(0.017)*	*(0.005)*	*(0.014)*	*(0.009)*	*(0.010)*	*(0.006)*

Notes: All results are conditional on the complete set of controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level.

Table 5: Effect of PGS and SES on Skills and Early Health

	Conscientiousness	Extraversion	Emotional Stability	Intelligence	Early Health
Education Polygenic Score	0.018	-0.002	0.009	0.122 ***	0.024
	0.019	0.019	0.014	0.020	0.018
Poor SES	-0.022	-0.050	-0.020	-0.138 ***	-0.055
	0.039	0.041	0.038	0.041	0.037
Education PGS x Poor SES	-0.034	0.002	-0.004	0.003	0.041
	0.034	0.036	0.028	0.037	0.033

Notes: All results are conditional on the complete set of controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level.

Table 6: Marginal Effects of PGS, SES, Skills, Health, and Interactions on Education

	Bachelor's Degree	High School Dropout
Education PGS	0.028 **	-0.006
	0.011	0.005
Poor SES	-0.046 **	0.025 **
	0.021	0.013
Education PGS x Poor SES	0.003	-0.001
	0.009	0.005
Conscientiousness	0.002	-0.007
	0.004	0.004
Extraversion	0.015 **	-0.009 *
	0.007	0.005
Emotional Stability	0.039 **	-0.018
	0.019	0.012
Intelligence	0.068 ***	-0.033 **
	0.024	0.016
Early Health	0.026 **	-0.005
	0.010	0.005
Early Health x Poor SES	0.001	-0.001
	0.010	0.005

Notes: All results are conditional on the complete set of controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level.

Table 7: Decomposition of the Direct Effect of the PGS on Health Outcomes

		Health (1)	Heavy Drinking (2)	Smoking Tobacco (3)	Marijuana Use (4)	Obese (5)	No Exercise (6)	Depression (7)	Suicidial Thoughts (8)
High SES									
	Health	0.006	0.000	0.000	0.000	-0.002	0.000	-0.001	0.000
		(0.004)	*(0.000)*	*(0.000)*	*(0.000)*	*(0.002)*	*(0.000)*	*(0.001)*	*(0.000)*
	Skills	0.006 *	-0.003 *	-0.004 **	0.000	-0.002	-0.001	0.000	-0.001
		(0.004)	*(0.001)*	*(0.002)*	*(0.001)*	*(0.001)*	*(0.001)*	*(0.002)*	*(0.001)*
	Unobserved channels	0.052 ***	-0.009	-0.026 **	-0.010 **	-0.010	-0.009	-0.019 **	0.006
		(0.018)	*(0.009)*	*(0.011)*	*(0.004)*	*(0.010)*	*(0.007)*	*(0.008)*	*(0.004)*
	Total	0.064 ***	-0.011	-0.031 ***	-0.010 **	-0.014	-0.010	-0.020 ***	0.004
		(0.019)	*(0.009)*	*(0.011)*	*(0.004)*	*(0.010)*	*(0.007)*	*(0.008)*	*(0.004)*
Low SES									
	Health	0.010 **	-0.001	-0.003 *	0.000	-0.003 *	-0.001	-0.002 *	0.000
		(0.005)	*(0.001)*	*(0.002)*	*(0.000)*	*(0.002)*	*(0.001)*	*(0.001)*	*(0.000)*
	Skills	0.006	-0.002	-0.003	0.000	-0.002	-0.001	0.000	-0.001
		(0.004)	*(0.002)*	*(0.002)*	*(0.001)*	*(0.001)*	*(0.001)*	*(0.002)*	*(0.001)*
	Unobserved channels	-0.038	0.007	-0.040 **	0.006	-0.015	0.011	0.000	-0.002
		(0.030)	*(0.013)*	*(0.016)*	*(0.005)*	*(0.015)*	*(0.009)*	*(0.010)*	*(0.006)*
	Total	-0.022	0.004	-0.047 ***	0.006	-0.019	0.010	-0.003	-0.004
		(0.031)	*(0.013)*	*(0.016)*	*(0.005)*	*(0.014)*	*(0.009)*	*(0.010)*	*(0.006)*

Notes: All results are conditional on the complete set of controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level.

Table 8: Decomposition of the Indirect Effect of the PGS on Health Outcomes

	Health	Heavy Drinking	Smoking Tobacco	Marijuana Use	Obese	No Exercise	Depression	Suicidial Thoughts
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
High SES								
Dropout	0.004 **	0.000	-0.004 ***	0.000	0.000	0.000	-0.001 *	0.000
	(0.002)	*(0.000)*	*(0.001)*	*(0.000)*	*(0.000)*	*(0.000)*	*(0.000)*	*(0.000)*
Bachelor's	0.014 ***	-0.007 ***	-0.016 ***	-0.005 ***	-0.005 **	-0.004 ***	-0.004 ***	0.001
	(0.004)	*(0.002)*	*(0.003)*	*(0.001)*	*(0.002)*	*(0.001)*	*(0.002)*	*(0.001)*
Total	0.017 ***	-0.007 ***	-0.019 ***	-0.005 ***	-0.005 **	-0.004 ***	-0.005 ***	0.000
	0.004	0.002	0.003	0.001	0.002	0.001	0.002	0.001
Low SES								
Dropout	0.000	0.000	-0.003 **	0.000 *	0.002 **	0.000	-0.001	0.000
	(0.001)	*(0.000)*	*(0.001)*	*(0.000)*	*(0.001)*	*(0.000)*	*(0.000)*	*(0.000)*
Bachelor's	0.029 ***	-0.006 *	-0.017 ***	-0.004 **	-0.005	-0.007 **	0.001	-0.001
	(0.009)	*(0.003)*	*(0.005)*	*(0.002)*	*(0.004)*	*(0.003)*	*(0.003)*	*(0.002)*
Total	0.029 ***	-0.006 *	-0.019 ***	-0.005 **	-0.003	-0.006 **	0.001	-0.001
	0.009	0.003	0.005	0.002	0.004	0.003	0.003	0.002

Notes: All results are conditional on the complete set of controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level.

4.3 Evidence for the Causal Effect of Education on Health

Table 9 shows the marginal effect of high school dropout and college completion on each health outcome. I find that higher thresholds of education are predictive of all health outcomes except for suicidal thoughts[13]. Dropping out of high school is associated with the direct effects lower general health (-27.8%) and a higher likelihood of smoking tobacco (28.2 %), marijuana use (2.4 %), and depression (6.9 %). I also find that college education is associated with every outcome other than suicidal thoughts, including a -19.8 % effect on smoking tobacco. Effects of college are all at least 5 % in magnitude.

A few interaction effects between education and SES are also found. These effects do not appear to substantially modify the overall positive effect of education, except the Bachelor's-SES interaction effect on depression and the dropout-SES interaction effect on health. These effects may simply be a function of family SES and education acting as substitutes in certain health production functions.

5 Conclusion

I find that an education polygenic score exhibits economically relevant effects on a variety of health outcomes. However, these effects are substantially moderated by SES, such that individuals who grew up in low-SES households do not experience the full health benefits of the education score. Genes are unlikely to be a policy variable in the foreseeable future due to ethical, political, and practical considerations, but I provide evidence that targeting the SES bottleneck may allow individuals to achieve greater health based on their genetic endowment. Based on the evidence here, targeting individuals at the low end of the SES spectrum appears to be an efficient way of boosting health outcomes. I contribute to an understanding of the mechanisms through which the PGS works and provide a mediation model that can be used to guide future research. I also provide

[13] A weak high school dropout-SES interaction exists for this outcome.

Table 9: Marginal Effect of Education on Health

	Health	Heavy Drinking	Smoking Tobacco	Marijuana Use	Obese	No Exercise	Depression	Suicidial Thoughts
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
High School Dropout	-0.278 ***	0.027	0.282 ***	0.024 *	-0.014	-0.001	0.069 **	0.021
	0.077	0.033	0.044	0.013	0.038	0.019	0.027	0.016
Dropout x SES	0.275 ***	-0.040	-0.088	0.008	-0.096 *	-0.028	-0.030	-0.041 *
	0.105	0.044	0.059	0.017	0.051	0.029	0.036	0.022
Bachelor's Degree	0.175 ***	-0.083 ***	-0.198 ***	-0.057 ***	-0.064 ***	-0.053 ***	-0.056 ***	0.006
	0.042	0.021	0.025	0.011	0.024	0.017	0.019	0.011
Bachelor's x SES	0.154 *	0.013	0.008	0.010	0.009	-0.023	0.069 **	-0.021
	0.085	0.041	0.050	0.023	0.045	0.034	0.034	0.022

Notes: All results are conditional on the complete set of controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level.

additional evidence that college education has a causal effect on health.

References

Aitken, M. and D. B. Rubin (1985). Estimation and hypothesis testing in finite mixture models. *Journal of Royal Statistical Society, Series B 47 (1)*, pp. 67–75.

Anderson, T. W. and H. Rubin (1956). Statistical inference in factor analysis. In *Proceedings of the third Berkeley symposium on mathematical statistics and probability*, Volume 5, pp. 111–150.

Barth, D., N. W. Papageorge, and K. Thom (2017). Genetic ability, wealth, and financial decision-making. Journal of Political Economy.

Belsky, D. W., T. E. Moffitt, D. L. Corcoran, B. Domingue, H. Harrington, S. Hogan, R. Houts, S. Ramrakha, K. Sugden, B. S. Williams, et al. (2016). The genetics of success: How single-nucleotide polymorphisms associated with educational attainment relate to life-course development. *Psychological science 27*(7), 957–972.

Bierut, L., P. Biroli, T. Galama, and K. Thom (2018). Childhood socioeconomic status moderates genetic predisposition for peak smoking. *bioRxiv*, 336834.

Boardman, J. D., B. W. Domingue, and J. Daw (2015). What can genes tell us about the relationship between education and health? *Social Science & Medicine 127*, 171–180.

Borghans, L., A. L. Duckworth, J. J. Heckman, and B. Ter Weel (2008). The economics and psychology of personality traits. *Journal of human Resources 43*(4), 972–1059.

Buckles, K., A. Hagemann, O. Malamud, M. Morrill, and A. Wozniak (2016). The effect of college education on mortality. *Journal of Health Economics 50*, 99–114.

Cameron, A. C. and P. K. Trivedi (2005). *Microeconometrics: methods and applications*. Cambridge university press.

Cangur, S. and I. Ercan (2015). Comparison of model fit indices used in structural equation modeling under multivariate normality. *Journal of Modern Applied Statistical Methods 14*(1), 14.

Conti, G., J. Heckman, and S. Urzua (2010). The education-health gradient. *American Economic Review 100*(2), 234–38.

Domingue, B. W., D. W. Belsky, D. Conley, K. M. Harris, and J. D. Boardman (2015). Polygenic influence on educational attainment: New evidence from the national longitudinal study of adolescent to adult health. *AERA open 1*(3), 2332858415599972.

Fletcher, J. M. (2019). Environmental bottlenecks in children's genetic potential for adult socio-economic attainments: Evidence from a health shock. *Population Studies 73*(1), 139–148.

Fletcher, J. M. and S. Schurer (2017). Origins of adulthood personality: The role of adverse childhood experiences. *The BE Journal of Economic Analysis & Policy 17*(2).

Freudenberg, N. and J. Ruglis (2007). Peer reviewed: Reframing school dropout as a public health issue. *Preventing chronic disease 4*(4).

Galama, T., A. Lleras-Muney, and H. van Kippersluis (2018, 05). The effect of education on health and mortality: A review of experimental and quasi-experimental evidence. *Oxford Research Encyclopedias: Economics and Finance*.

Grossman, M. (2017). *The demand for health: a theoretical and empirical investigation*. Columbia University Press.

Harris, K. M. and D. B. Braudt (2018). Polygenic scores (pgss) in the national longitudinal study of adolescent to adult health (add health)–release.

Heckman, J. and B. Singer (1984). A method for minimizing the impact of distributional

assumptions in econometric models for duration data. *Econometrica: Journal of the Econometric Society*, 271–320.

Heckman, J. J. (2008). Schools, skills, and synapses. *Economic Inquiry 46*(3), 289–324.

Idler, E. L. and Y. Benyamini (1997). Self-rated health and mortality: a review of twenty-seven community studies. *Journal of health and social behavior*, 21–37.

Lee, J. J., R. Wedow, A. Okbay, E. Kong, O. Maghzian, M. Zacher, T. A. Nguyen-Viet, P. Bowers, J. Sidorenko, R. K. Linnér, et al. (2018). Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals. *Nature genetics 50*(8), 1112.

Marioni, R. E., S. J. Ritchie, P. K. Joshi, S. P. Hagenaars, A. Okbay, K. Fischer, M. J. Adams, W. D. Hill, G. Davies, R. Nagy, et al. (2016). Genetic variants linked to education predict longevity. *Proceedings of the National Academy of Sciences 113*(47), 13366–13371.

Mroz, T. A. (1999). Discrete factor approximations in simultaneous equation models: Estimating the impact of a dummy endogenous variable on a continuous outcome. *Journal of Econometrics 92*(2), 233–274.

Papageorge, N. W. and K. Thom (2018). Genes, education, and labor market outcomes: evidence from the health and retirement study. Technical report, National Bureau of Economic Research.

Price, A. L., N. J. Patterson, R. M. Plenge, M. E. Weinblatt, N. A. Shadick, and D. Reich (2006). Principal components analysis corrects for stratification in genome-wide association studies. *Nature genetics 38*(8), 904.

Savelyev, P. and K. T. Tan (2017). Socioemotional skills, education, and health-related outcomes of high-ability individuals. *American Journal of Health Economics* (Just Accepted), 1–73.

Schafer, J. L. (1999). Multiple imputation: a primer. *Statistical methods in medical research 8*(1), 3–15.

Solovieff, N., C. Cotsapas, P. H. Lee, S. M. Purcell, and J. W. Smoller (2013). Pleiotropy in complex traits: challenges and strategies. *Nature Reviews Genetics 14*(7), 483.

Strauss, J. and D. Thomas (2007). Health over the life course. *Handbook of development economics 4*, 3375–3474.

Young, J. K. and A. A. Beaujean (2011). Measuring personality in wave i of the national longitudinal study of adolescent health. *Frontiers in psychology 2*, 158.

Yung, Y.-F. and K.-H. Yuan (2013). Bartlett factor scores: General formulas and applications to structural equation models. In R. E. Millsap, L. A. van der Ark, D. M. Bolt, and C. M. Woods (Eds.), *New Developments in Quantitative Psychology*, New York, NY, pp. 385–401. Springer New York.